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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER
WILSON, MICHAEL C

ART UNIT	PAPER NUMBER
1632	19

DATE MAILED: 07/30/2002

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No. 09/087,513	Applicant(s) KANEKO ET AL.	
	Examiner Michael Wilson	Art Unit 1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *15 July 2002* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *14,15,19 and 21-27* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *14,15,19 and 21-27* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☒ Other: *detailed action* .

DETAILED ACTION

The Art Unit location of your application in the USPTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Art Unit 1632.

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 7-5-02, paper number 18, has been entered.

The amendment filed 2-5-02, paper number 15, has been entered. Claim 20 has been canceled. Claims 21-27 have been added. Claims 14, 15, 19 and 21-27 are pending and under consideration in the instant application. Applicant's arguments filed 2-5-02, paper number 15, have been fully considered but they are not persuasive. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Specification

Applicants have returned page 26, line 17, to its original state. As such, the objection to the specification has been revived:

The specification refers to a vv-ΔV3 mutant with the Δ297-329 deletion in 15 incorporated by reference herein in its entirety (page 26, line 17). However, reference 15 is not

cited such that the reference can be determined. Deletion of "15, incorporated by reference herein in its entirety" is suggested.

Claim Rejections - 35 USC § 112

The objection to the amendment filed 4-11-01 under 35 U.S.C. 132, because it introduces new matter into the disclosure on page 26, line 17, is withdrawn because of the deletion of Wyatt.

1. Claims 21, 24, 26 and 27 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The phrase "deletion of amino acids 297-329 in said variable loop" is new matter. The specification does not give any indication that the deletion is an amino acid deletion and not a nucleic acid sequence deletion. Nor is it readily apparent to one of skill in the art at the time of filing that the deletion is of amino acids. As such, the phrase is new matter.

2. Claims 14, 15 and 19 remain rejected and claims 21-27 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention for reasons of record.

The claims require DNA encoding an envelope glycoprotein of HIV having a deletion of the third variable loop. The only DNA disclosed in the specification having a deletion of the V3 loop as claimed are vv-ΔV3 (pg 26), 1△V3, 7△V3 and 8△V3 (pg 34). The specification does not

provide adequate written description for one of skill to make the DNA disclosed in the specification having a deletion of the V3 loop.

The specification does not provide adequate written description for vv-ΔV3. Kmieciak et al. (June 1, 1998, J. Immunol., Vol. 160, pg 5676-5683) teaches vv-ΔV3 was made using "the Δ297-329 deletion" taught by Wyatt (Dec. 1992, J. Virology, Vol. 66, pg 6997-7004). The Δ297-329 deletion of Wyatt was a deletion spanning the V3 loop, wherein Gly-Ala-Gly was inserted in place of the loop. Wyatt also taught the strain of HIV used to make the Δ297-329 deletion was HXBc2 (pg 6998, col. 1, 2nd para.). The specification does not teach the Δ297-329 deletion had Gly-Ala-Gly inserted in its place. The specification does not teach the starting material was HXBc2. In fact, the specification teaches the strain was HIVIIIB. Nor does the specification reference Wyatt. The specification does not teach which nucleic acids are 297-329 and such nucleotides cannot be determined from the art. Are the numbers describing the V3 loop or the HIV IIIB genome? Did nucleotides 297-329 have an art accepted meaning at the time the invention was made? As such, the specification does not provide adequate written description for one of skill to make vv-ΔV3.

Applicants argue the specification teaches how to make vv-ΔV3 and points to page 26, line 16, through page 27, line 14. Applicants argument is not persuasive. The specification states HIV-IIIB mutants with the ▵297-329 deletion were made by ligation of fragments obtained by PCR from pSCIII-env. The specification does not teach the Δ297-329 deletion had Gly-Ala-Gly inserted in its place. The specification teaches the starting material was HIV IIIB, not HXBc2.

The specification does not provide any indication that the ligation of the fragments disclosed results in the Δ297-329 deletion and the insertion of 3 amino acids in its place as required in vv-ΔV3 (Kmieciak).

Applicants argue the method used to make vv-ΔV3 disclosed in the specification is also disclosed in Kmieciak et al. (June 1, 1998, J. Immunol., Vol. 160, pg 5676-5683). Applicants argument is not persuasive because Kmieciak was not available at the time of filing (May 29, 1998).

The specification does not provide adequate written description for the 1△V3, 7△V3 and 8△V3 mutants. The specification discloses the 1△V3, 7△V3 and 8△V3 mutants in Example 14 (page 34, Fig. 1) but does not teach how to make such mutants, how the mutants differ from each other, how the mutants differ from the vv-△V3 mutant with the △297-329 deletion (page 26) or the structural elements of the mutants. The specification discloses the WTP-2, WTP-5 and WTP-8 (page 35, line 3; page 36, line 16; Fig. 1), but it is unclear how the envelope gene in these vectors differs from each other or from the V3 mutants or whether these vectors are considered "modified".

Applicants argue the 1△V3, 7△V3 and 8△V3 mutants are different clones obtained by homologous recombination as described on pg 27, line 12 and pg 34, line 19, and pg 35, line 1. Applicants conclude the 1ΔV3 mutant is the first clone obtained after homologous recombination, the 7ΔV3 is the seventh clone obtained after homologous recombination. Applicants arguments are not persuasive. Example 14 (pg 34, line 19) does not teach the mutants were made using

homologous recombination as taught on pg 27, line 12. The structure of the mutants cannot be determined and the difference between the "first clone" and "seventh clone" cannot be determined.

Applicants argue WTP-2, WTP-5 and WTP-8 are wild-type vectors that do not have a deletion of the V3 loop as claimed. While pg 27, line 10 states, pSC-WTP have the "WT env gene", the specification does not teach how the env is modified. Thus, it remains unclear how WTP-2, WTP-5 and WTP-8 correlate to DNA having a deletion of the V3 loop as claimed.

Claims 14, 15, require the product made is a vaccine against HIV. Claims 19 require the product is a vaccine that induces cellular immunity against HIV. The only disclosed purpose for vaccinating against HIV or inducing cellular immunity against HIV is to treat or prevent HIV infection (pg 1, line 12; pg 23, line 9). The specification does not provide adequate written description for any DNA encoding an envelope glycoprotein of HIV with a deletion in V3 capable of treating or preventing HIV, specifically capable of inducing a cellular immune response against HIV that is therapeutic or prophylactic.

The specification does not provide adequate written description for DNA encoding an HIV envelope glycoprotein having a deletion in the V3 loop or a cell expressing such an envelope glycoprotein that is capable for being used to treat or prevent HIV, specifically to induce a therapeutic or prophylactic cellular immune response against HIV. The specification does not teach obtaining a cellular immune response that is directed toward HIV or obtaining a therapeutic or prophylactic effect against HIV using the DNA or cells as claimed. The art at the time of filing

did not teach how to obtain such an effect using DNA encoding an HIV envelope protein having a deletion in the V 3 loop. Without such guidance, the specification does not provide adequate written description for the structure of any DNA encoding an envelope glycoprotein of HIV with a deletion in V3 having the function of treating or preventing HIV.

Applicants argue pg 41, line 21, through pg, 42, line 7, describes vaccinating with ΔV3 mutants "which will also reduce the need for immunization with HIV staring-specific env glycoproteins," delivering the mutants as plasmid DNA or cells. In addition, applicants argue the specification teaches ΔV3 mutants interact with anti-gp120 antibodies of HIV patients suggesting that the mutants are capable of inducing antibody responses *in vivo*. Applicants arguments are not persuasive. The suggestion to use the DNA as a vaccine, either plasmid or cells transfected with the DNA, is not adequate written description for the DNA actually having the ability to treat or prevent HIV. A mere suggestion that the mutants are capable of inducing antibody responses *in vivo* is not adequate to indicate the DNA actually treats or prevents HIV in view of the dearth of evidence in the specification or the art at the time of filing regarding the use of DNA encoding HIV env having a deletion of V3 for the treatment or prevention of HIV. Furthermore, "interactions" between ΔV3 mutants and antibodies does not indicate the antibodies are useful in treating or preventing HIV. Finally, an antibody immune response is not a cellular immune response as in claims 19, 23, 27 or used when APCs express the ΔV3 mutants (claim 15, 23).

3. Claims 14, 15 and 19 remain rejected and claims 21-27 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a

way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention for reasons of record.

The claims require DNA encoding an envelope glycoprotein of HIV having a deletion of the third variable loop. The only DNA disclosed in the specification having a deletion of the V3 loop as claimed are vv-ΔV3 (pg 26), 1ΔV3, 7ΔV3 and 8ΔV3 (pg 34). The specification does not enable one of skill to make the ΔV3 mutants disclosed in the specification.

The specification does not enable one of skill to make vv-ΔV3. Kmieciak et al. (June 1, 1998, J. Immunol., Vol. 160, pg 5676-5683) teaches vv-ΔV3 was made using "the Δ297-329 deletion" taught by Wyatt (Dec. 1992, J. Virology, Vol. 66, pg 6997-7004). The Δ297-329 deletion of Wyatt was a deletion spanning the V3 loop, wherein Gly-Ala-Gly was inserted in place of the loop. Wyatt also taught the strain of HIV used to make the Δ297-329 deletion was HXBc2 (pg 6998, col. 1, 2nd para.). The specification does not teach the Δ297-329 deletion had Gly-Ala-Gly inserted in its place. The specification does not teach the starting material was HXBc2. In fact, the specification teaches the strain was HIVIIIB. Nor does the specification reference Wyatt. The specification does not teach which nucleic acids are 297-329 and such nucleotides cannot be determined from the art. Are the numbers describing the V3 loop or the HIV IIIB genome? Did nucleotides 297-329 have an art accepted meaning at the time the invention was made? As such, the specification does not enable one of skill to make vv-ΔV3.

The specification does not enable one of skill to make 1ΔV3, 7ΔV3 or 8ΔV3. The specification discloses the 1ΔV3, 7ΔV3 and 8ΔV3 mutants in Example 14 (page 34, Fig. 1) but

does not teach how to make such mutants, how the mutants differ from each other, how the mutants differ from the vv-ΔV3 mutant with the Δ297-329 deletion (page 26) or the structural elements of the mutants. The specification discloses the WTP-2, WTP-5 and WTP-8 (page 35, line 3; page 36, line 16; Fig. 1), but it is unclear how the envelope gene in these vectors differs from each other or from the V3 mutants or whether these vectors are considered "modified".

Claims 14, 15, 21, 22 and 26 require the product made is a vaccine against HIV. Claims 19, 23-25 and 27 require the product induces cellular immunity against HIV. The only disclosed purpose for vaccinating against HIV or inducing cellular immunity against HIV is to treat or prevent HIV infection (pg 1, line 12; pg 23, line 9). The specification does not enable one of skill to use DNA encoding ΔV3 mutants to treat or prevent HIV.

At the time of filing, it was unpredictable whether a nucleic acid construct would have a therapeutic or prophylactic effect against HIV. Ross of record (September 1996, Human Gene Therapy, Vol. 7, pages 1781-1790) states a major technical impediment to gene transfer is the lack of ideal gene delivery systems including vectors, promoters and modes of delivery (page 1782, column 2, first full paragraph). These technical parameters are required to obtain efficient delivery and sustained expression of the gene (Verma of record, Sept. 18, 1997, Nature, Vol. 389, page 239-242; see page 239, 3rd column, line 10). The difficulties in sustaining expression of a gene cause an unpredictability in obtaining a therapeutic or prophylactic effect in a patient (Ross, page 1789, column 1, first paragraph). Therefore, the parameters required to obtain a therapeutic effect using DNA were unpredictable at the time of filing.

Regarding vaccines, it was unpredictable how to obtain a therapeutic effect against a virus using a single antigenic stimulus as a vaccine. Haynes of record (1993, Science, Vol. 260, pages 1279-1286) teaches the classic approach to vaccine development involves exposing cells of the immune system to the proper antigenic stimulus which stimulates a beneficial immune response. The prior art presents few examples where a single antigenic stimulus, such as a small limited peptide or a whole protein is found to engender a therapeutic or protective immune response. The successful art-recognized immunogens used as vaccines are derived from whole killed or live attenuated pathogens, comprised of complex antigenic mixtures or comprised of inactivated toxins. Many of these successes were achieved with a certain degree of luck, influenced by some particular peculiarity or aspect of a given pathogenic agent. Therefore, it was unpredictable how to obtain a therapeutic effect against a virus using a single antigen.

Specifically regarding HIV vaccines, Stricker of record (Medical Hypotheses, June 1997, Vol. 48, pages 527-9; see page 527, last paragraph through all of page 528) teaches that attempts to develop a vaccine against HIV have been unsuccessful. In fact, HIV infection has defied the creation of an effective vaccine or immunotherapeutic. Overall, a lack of understanding about cellular immunity against HIV, the sequence variability of HIV and the rapid replication of HIV, as disclosed by Bangham of record contribute the ineffectiveness of vaccines against HIV (Nov. 29, 1997, Lancet, Vol. 350, pages 1617-1621; page 1617, top of column 1). It is not known what renders an antigen capable of stimulating beneficial or protective CTL responses to HIV. Therefore, the art at the time of filing did not teach that the envelope glycoprotein of HIV could

be used to induce a therapeutic cellular immune response against HIV. Thus, the parameters required to obtain a therapeutic cellular immune response against HIV was unpredictable at the time of filing.

The specification does not enable one of skill in the art to use DNA encoding ΔV3 mutants or a cell expressing ΔV3 mutants for treatment or prevention HIV. The specification does not teach obtaining a cellular immune response that is directed toward HIV, obtaining a therapeutic or prophylactic immune response against HIV using DNA encoding ΔV3 mutants, specifically obtaining a therapeutic or prophylactic cellular immune response against HIV using DNA encoding ΔV3 mutants. The art at the time of filing did not teach how to obtain such an effect using DNA encoding ΔV3 mutants. Without such guidance, the specification does not enable one of skill in the art to use DNA encoding ΔV3 mutants to treat or prevent HIV.

Applicants arguments regarding the enablement issues described above are addressed in the response to the arguments regarding the written description rejection.

The specification provides CTL and antibody-dependent cell-mediated cytotoxicity data *in vitro* (page 35-38), but does not provide any examples of inducing cellular immunity against HIV *in vivo*. Nor does the specification provide adequate correlative evidence between *in vitro* data and *in vivo* results such that a therapeutic cellular immune response against HIV could be obtained *in vivo*.

The state of the art at the time of filing was that CTL assays *in vitro* produce variable results depending on the target cells used, the effector to target ratio used, and the incubation

time (Lancki of record, 1992, Biotherapy, Vol. 5, pages 71-81; see page 72, column 1, line 1) CTL assays combine PBL and target cells that are artificially "loaded" with antigen. The amount of antigen required on the target cell surface to induce a CTL response depends upon the immunostimulatory epitope of the antigen, the type of immune response and the strength of the immune response desired. Moreover, CTL assays do not account for the complex interaction of the immune response and cytokine regulation that occurs *in vivo*. For example, Bachmann of record reviews the use of the cellular immune response both *in vivo* and *in vitro* in viral assay systems (1994, Current Op. Immunol. Vol. 6, pages 320-326). A comparison of sensitivities shows that radioactive CTL assays are more sensitive than *in vivo* assays, but that results of secondary *in vitro* stimulation need to be verified by *in vivo* assay. On page 323, Bachmann states one should be very cautious not to 'over-interpret' results obtained by a cytolytic assay where cells are stimulated *in vitro* because the results may be biologically irrelevant without *in vivo* confirmation. Therefore, it was unpredictable at the time of filing whether a CTL response obtained *in vitro* could be obtained *in vivo* or that a cellular immune response obtained *in vivo* equivalent to the cellular immune response obtained *in vitro* will have any biological relevance.

The *in vitro* CTL and ADCC assays disclosed in the instant application require PBMC isolated from an HIV patient and autologous B-LCL or Jurkat cells transfected with the vectors of the invention as target cells which do not correlate to cells or nucleic acids used to treat viral infection *in vivo*. The specification does not teach the strain of HIV in the patients used to make the PBMC *in vitro*, the level of antigen expression on the surface of target cells *in vitro*, the level

of expression required *in vivo*, or how the immune response obtained *in vitro* correlates to response expected *in vivo*. It is not clear that the ratios of target to effector ratio used *in vitro* correlates to the ratio of transfected cells to effector cells that would occur *in vivo*. It addition, applicants activated the PBMC with antibodies which is an artificial means used to increase the activity of the cytotoxic cells and does not correlate to conditions found in the HIV patients because patients PBMCs are not stimulated with anti-CD3 antibodies. In addition, the specification does not teach that the level of cellular immunity *in vitro* would have any therapeutic benefit in a patient. Given the state of the art regarding the lack of correlation between *in vitro* and *in vivo* cytotoxicity taken with the guidance provided in the specification, it would have required one of skill undue experimentation to determine the parameters required to obtain an cellular immune response *in vivo* that has a therapeutic or prophylactic effect.

Applicants argue that *in vitro* CTL data correlates with *in vivo* protective efficacy as supported by the manuscript by Kiszka. Applicants argument is not persuasive. Applicants have provided no nexus between the ΔV3 mutant of Kiszka and the ΔV3 mutant disclosed in the instant application, between *in vitro* disclosed in the specification and *in vivo* CTL responses obtained in Kiszka, between increasing resistance to vaccinia virus transmission and HIV transmission. Kiszka does not indicate the CTL response was therapeutic or prophylactic against HIV; the mice were challenged with vaccinia encoding HIV gp160, not HIV.

Claims 14, 15, 19, 21, 23 and 24 encompass modifying the envelope glycoprotein of any strain of HIV. The state of the art at the time of filing was such that the V3 region of HIV varied

between HIV strains and mutated frequently (page 1, line 15; page 2, line 13; page 3, line 3). The specification only teaches modifying the V3 loop of the HIV-IIIB envelope glycoprotein (page 26, line 12). The specification does not teach how to modify the V3 loop of the envelope glycoprotein of any other strain of HIV or correlate the V3 loop of HIV-IIIB to other strains of HIV such that similar modifications could be made or that a therapeutic cellular immune response could be induced against the glycoprotein.

Applicants demonstrate different modifications of HIV-IIIB cause different effects (e.g. 1ΔV3, 7ΔV3 or 8ΔV3 mutants induce different immune responses, Fig. 1). Therefore, the specification does not enable one of skill to determine how to modify the V3 loop of any HIV envelope glycoprotein such that a therapeutic cellular immune response against HIV is obtained.

Overall, the specification does not provide adequate guidance regarding how to induce a therapeutic cellular immune response against HIV *in vivo*. Given the state of the art taken with the guidance provided in the specification, it would require one of skill undue experimentation to determine how to use DNA encoding ΔV3 mutants to induce a therapeutic or prophylactic cellular immune response against HIV.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

4. Claims 21, 24, 26 and 27 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

It is unclear from the specification that the deletion of the V3 loop is of amino acids 297-329 as claimed and not of nucleotides 297-329 in the DNA.

Claims 14, 15, 21, 22 and 26 appear to be free of the prior art of record because the prior art of record did not teach or suggest combining DNA encoding a ΔV3 mutant with adjuvant. Claims 19, 23-25 and 27 appear to be free of the prior art of record because the prior art of record did not teach or suggest combining cells expressing DNA encoding a ΔV3 mutant with adjuvant.

Conclusion

No claim is allowed.

Inquiry concerning this communication or earlier communications from the examiner should be directed to Michael C. Wilson who can normally be reached on Monday through Friday from 9:00 am to 5:30 pm at (703) 305-0120.

Questions of formal matters can be directed to the patent analyst, Dianiece Jacobs, who can normally be reached on Monday through Friday from 9:00 am to 5:30 pm at (703) 305-3388.

Questions of a general nature relating to the status of this application should be directed to the Group receptionist whose telephone number is (703) 308-1235.

If attempts to reach the examiner, patent analyst or Group receptionist are unsuccessful, the examiner's supervisor, Deborah Reynolds, can be reached on (703) 305-4051.

The official fax number for this Group is (703) 308-4242.

Michael C. Wilson

MICHAEL C. WILSON
PATENT EXAMINER